Reply

In reply to the Letter by T. Menovsky, we would first agree with his general comments in support of laser-activated nerve repair as a technique with significant advantages of faster repair time, no long-term foreign body reaction and less scarring at the repair site. In addition, we believe that the use of an applied protein solder is helpful both in strengthening the bond formed between the tissues and in protecting the underlying tissues from thermal damage. We have found that our solid protein solder is easy to apply and allows a controlled and reproducible laser energy dose to the protein solder, which is helpful in ensuring thorough coagulation of the protein solder, without excessive heating.

Figure 3 in our paper shows a light micrograph of nerve tissue immediately after laser-protein-solder repair. It shows that some axons in a region up to ~100 μm below the laser-treated solder and perineurium are in transverse section, possibly due to severing and manipulation during the operation. (We have also observed similar twisting of axons after suturing [1], which does not involve any thermal effect). In this localised region, there is evidence of edema in the axons, which is attributable to thermal damage, although there is no evidence of thrombosed vessels. (Note that we did not perfuse the tissues before histological preparation). Because of the localised heating induced by the laser light being absorbed in the protein solder, it is probably inevitable that there is some heat transfer to the underlying tissue. In particular the nerve of Figure 3 had no perineurium at the repair site, (as correctly noted by Menovsky), possibly due to untrimmed axon extrusion after severing, and that is expected to increase the localised thermal damage of the axons in contact with the solder. However, since the underlying tissue does not directly absorb the laser light, this heat is localised to the region immediately adjacent to the laser-treated protein solder and is possibly more pronounced in the figure cited than that shown in earlier studies [1].

In subsequent work (McNally et al., unpublished observation), we have noted the importance of controlling the laser energy dose, so as not to overheat the protein solder. (In this case the laser energy dose was controlled by reference to the surface temperature reached by the protein solder during irradiation). We believe that our results in this subsequent study offer the prospect of further reducing thermal damage to underlying tissue by optimising the energy delivery to the laser-activated protein solder.

We adopted a nerve repair protocol involving the resection of the epineurium (normally with trimming of the extruded axons) as we anticipated that the resultant ability to perform fasicular repair would be more satisfactory for long-term axon regeneration and continuity than that obtained when the epineurium is retained. Despite some (localised) thermal damage observed in these specimens, the long-term (3 months) recovery results are very encouraging.

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REFERENCE

1. Trickett R, Lauto A, Dawes JM, Owen ER. Laser activated protein solder for peripheral nerve repair. Proc SPIE 1995; 2395:542–546.

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